Rheumatic Purpura.

1883. 12.

by

Dr Wickham Legg.

(Author's copy presented to Dr W. Bulloch. 1909.)

J.W. Hayward, Lancet, II. 54. 1883. Malignant Scarlatina treated by Crotalus.

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Augusto Murri Intorno al contagio scorbutico (cuttings & pamphlets) bound (left side)

Man, among skins, potter: July 11. measles & hæmorrhagic purpura of legs: no microbes in blood
July 23. Rash purpuric on legs: Discharging arms
Man, Luke, July 9. syphilis & purpura: no microbes

Guelliot (Union méd du Nord-Est) quoted in Journal de Méd. 1884 avril p. 164. Urine albumen in same then convulsions vomiting coma; second to these purpuric spots on mucous & internal skin of thighs (mulberry rash of English) Death in 20 hours

Heynlschak Infective purpura Arch. f. Kinderheilkunde 1884. p. 461. Bd. V.

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Man, Luke, July 9. syphilis & purpura: no microbes

[Reprinted from St. Bartholomew's Hospital Reports, Vol.

A CASE OF RHEUMATIC PURPURA,

WITH NOTES.

BY

J. WICKHAM LEGG, M.D.

By purpura we mean the appearance of numerous spontaneous hæmorrhages, either under the skin or from the mucous membranes; in short, the state which the old French pathologists would have called a hæmorrhagic diathesis.

I suppose no one would be inclined to maintain that this purpura is a pathological entity, due always to the same cause. It is seen in such a multitude of diseases, that, before dealing with the following case, it may be well to put side by side the different states in which purpura may be found.

(i.) The acute purpura which is seen in eruptive diseases. This proves rapidly fatal, and often ends in less than forty-eight hours. Sydenham noticed the hæmorrhagic form of both smallpox and the plague,[1] and how fatal it was. Cornil has examined very carefully the pustules in hæmorrhagic variola, but it can hardly be said that he has made out more than that the corpuscles escape in surprising abundance into the rete mucosum; he inclines to an escape *per diapedesin*.[2]

The cause of the infective purpura is, of course, set down by the extreme supporters of the germ theory to the presence of bacteria. Ceci even names one germ the *Monas hæmorrhagicum*, an organism which he has found in patients who have died from hæmorrhagic smallpox.[3] It may be well not to forget in these cases the influence of the high temperature. Bouchard put a dog into a warm bath, so as to keep his temperature at 44° C., and found that he had caused ecchymoses of the tissue of the heart.[4] The parenchymatous degeneration of the glands

[1] Thomas Sydenham, Obs. Med. iii. 2, § 24, ed. Greenhill, p. 128.

[2] Cornil, Bulletins de la Société méd. des Hôpitaux de Paris, 1879, p. 322.

[3] Ceci, Arch. f. exp. Path. 1881, Bd. xiii. p. 641.

[4] Bouchard, Comptes rendus des Séances de la Société de Biologie, 1870, p. 27.

and muscles caused by fever appears to be the same as that caused by the agents spoken of in the next paragraph but one, (iii.,) phosphorus, alcohol, and others.

Purpura in typhoid fever is thought to be a dangerous complication, and nearly all the cases recorded have proved fatal. Schneschkow has, however, lately published a case in which purpura appeared on the sixteenth day of typhoid fever in a child of six; the attack was severe, bleedings taking place from the nose and mouth, under the skin, with the urine and stools; pains were felt in the right knee. On the 21st day of the fever the bleedings ceased, and the patient made a good recovery.[1]

Some years ago I was asked to see a little girl suffering from purpura and epistaxis during the progress of whooping-cough. The patient did well. I see that a case of the same kind has been lately recorded in Italy. But Walker, so long ago as 1797, printed a case of a hæmorrhagic diathesis coming on and disappearing during an attack of whooping-cough.[2]

(ii.) Next to the infective, we may put the toxic, purpura, which follows the action of so many drugs and poisons, including snake-bites.[3] It would be a hard matter to make a complete enumeration of the drugs which cause hæmorrhage as part of their physiological action. That this was a property of iodide of potassium and bromide of potassium has long been known. Ordinary kitchen-salt also possesses this power, and the mode in which it acts has also been made out. It causes hæmorrhage *per diapedesin*. Prussak injected a 2 *per cent.* solution of chloride of sodium into the lymph sac of frogs, and found that the red corpuscles began to escape through the uninjured vessels, and to be visible in the tissues around. He also injected daily a certain quantity of chloride of sodium into the connective tissue of a rabbit, and after death numerous ecchymoses were found in all the organs.[4] The experiment on the frog I repeated many times with success in 1871. A crystal of chloride of sodium was put under the skin, and the circulation in the web of the foot watched under the microscope. The red corpuscles were seen applying themselves to the sides of the capillaries, escaping through the wall, and they became visible in the tissues

[1] Schneschkow, Medicinskoje Obosrenije, 1881, Feb. abstract in Centralblatt f. klin. Med. Bd. ii. 1882, p. 163.

[2] Walker, Duncan's Annals of Medicine for 1797, vol. ii. p. 231.

[3] Dr. Weir Mitchell thinks the hæmorrhage in snake-bites and in yellow fever to be due to the weakened vessels. (American Journal of the Medical Sciences, 1869, vol. lviii. p. 120.)

[4] Prussak, Sitzungsberichte der math.-naturw. Classe der kaiserl. Akad. der Wissenschaften, Wien, 1867, Bd. lvi. Abth. ii. p. 13.

Diabetes said to be often complicated with purpura: a case under Andrew in Mark, May 1884. Schoolmaster.
Blood examined.

Pregnancy complicated with haemor. Schuma in Jahresb. f. 1883. Bd. ij. p.

Scurvy: Cerebral haemorrhage. Frend, Wiener med. Woch. 1884. 9. 410. Cent. f. k. M. 1884. 374

Toxic purpura? Man in Mark soon after Xmas 1883. Who kept meat till it was high ate it & then had purpura

Purpura after rat bite Union méd. 1884. No 183

Epistaxis in cirrhosis Heberden Chap. Ch 64. p 323

around. This escape of the corpuscle would be either entire or in part. In the latter case, after, say, one-half of the corpuscle had made its way through the wall of the vessel, the remainder would stay attached to the wall, but at length be washed away and disappear in the general circulation. At the same time I also made a number of observations upon the action of iodide of potassium and bromide of potassium, with other alkaline chlorides, iodides, and bromides, thinking that they too might cause the same hæmorrhage *per diapedesin* as the chloride of sodium; but I saw no such escape of the red corpuscles after their action. And I may say that the observations were all made at the same time of the year (I fancy in the spring) both the successful and the unsuccessful ones.[1]

(iii.) From this toxic purpura we descend by easy stages to the purpura of disease—stages so easy that it is hard to say where the toxic influence ends and that of disease begins. In a paper published in these Reports some five years ago,[2] I endeavoured to bring under one head, to which I gave the name *Icterus gravis*, several varieties of disease, some caused by drugs, others idiopathic. These states have many common symptoms, one of the most prominent of which is purpura, and the appearances seen after death are so alike, that it would be hard even for an accomplished morbid anatomist to distinguish them. Such are chronic poisoning by phosphorus, arsenic, antimony, chloroform, chloral, alcohol, lead, silver, and many other drugs, yellow fever, acute yellow atrophy of the liver, and what is called bilious typhoid. In all these it is possible that the cause of the purpura may be the degeneration of the vessels, in itself only a part of the wide-spread cloudy swelling, or parenchymatous degeneration of the glands and muscles of the body, like that caused by febrile high temperatures.

After these come the purpura of anæmia, allied to the foregoing in the fatty degeneration of the tissues, the purpura of Bright's disease and of cirrhosis, not forgetting the hæmorrhages so often seen when a case of long-continued jaundice is about to end fatally. Three years ago Dr. Gee called attention to the presence of purpura in cases of chronic nephritis;[3] (and I have now under my care a case of purpura accompanying well-marked Bright's disease); some persons, it is well known, look upon Bright's disease as one of the manifestations of long-continued intoxication with alcohol. So also in cirrhosis. In the early stages of cirrhosis purpura is very common. I think that hardly sufficient atten-

[1] I published the results in 1872 in my Treatise on Hæmophilia, p. 91, note.
[2] St. Bartholomew's Hospital Reports, 1878, vol. xiv. p. 43.
[3] Samuel Gee, St. Bartholomew's Hospital Reports, 1880, vol. xvi. p. 47.

tion has been called to the repeated epistaxis, the spongy gums, and the purpuric spots of early cirrhosis. Sometimes the hæmorrhages are noticed earlier than the hepatic symptoms. There has been under my care quite lately in Luke's Ward a man, by occupation a cellarman, who was sent to me on account of purpura on the limbs, epistaxis, and swollen gums; the large liver and history of the abuse of wine not having been noted. The hæmorrhages in cirrhosis are sometimes explained when they occur in the portal tract by attributing them to congestion. This does not, however, explain hæmorrhages in parts far distant, like the limbs and gums. I would rather suggest that as hæmorrhage is one of the results of an acute poisoning by alcohol, so it may also follow a slow poisoning, and that the purpura is only one sign of the action of alcohol on the body, of which the chief damage falls upon the liver. The swelling of the spleen in cirrhosis I take to be also a part of the general disorder, not due solely to the congestion. So in Bright's disease, often due to some poison, the purpura may be part of the general action of the drug, of which the nephritis is a local expression. In long-continued jaundice, purpura is common as the jaundice is about to prove fatal, and it is possible that this purpura follows the long-continued circulation of the bile in the body.

(iv.) There comes next a large number of diseases in which purpura is noticed, but in which even a speculative explanation is wanting. For example, agues, of which paroxysmal hæmaturia is an instance, a disease which has lately been much studied, of which apparently Hippocrates spoke when he described the cold as doing harm to those who make bloody water in cold weather.[1] Professor Porter, in a paper communicated to the Medico-Chirurgical Society by Mr. Marsh, has described traumatic hæmorrhages as the result of ague.[2] Syphilis, phthisis, and general tuberculosis are sometimes attended by purpura.

(v.) Purpura due to congestion may next be spoken of. It appears to be a form more readily explained than the others. Cohnheim, after tying the vein which arose from some transparent part, as the web of the foot or the tongue, noticed, among other phænomena, that the red corpuscles began to escape in great numbers through the wall of the apparently uninjured vessel,[3] a true hæmorrhage *per diapedesin*, such as I have myself observed after the action of chloride of sodium. Thus

[1] Hippocrates, De Liq. Usu, cap. 6, Littré's ed. t. vi. p. 134.
[2] Porter, Med. Chir. Trans. 1876, vol. lix. p. 136.
[3] Cohnheim, Arch. f. path. Anat. 1867, Bd. xli. p. 220.

Finch, Lancet. 1880. Vol. ij. p. 557.
Fiedet, Union méd. 1880. N° 36.

Clark, Splenic ? Purpura, Lancet May 24. 1884. No bacteria in blood

Petersen, Syphilis haemorrhagica neonatorum Vierteljahrsschr. f. Derm. u. Syph. 1883. p. 509.

the purpura seen in the lower limbs in heart-disease is best explained.

(vi.) In former times one of the favourite ways of explaining constitutional hæmorrhage was by invoking a change in the blood. It has already been said that purpura accompanies anæmia; and it is not always that the loss of blood is the cause of the anæmia, though this appears to be very commonly the case. One result of hæmorrhage is an increase of the colourless blood corpuscles; and an increase in the colourless corpuscles seems, in its turn, to be often a cause of hæmorrhage. How common purpura and epistaxis are in leucæmia need not be noticed. The white corpuscles form masses or balls very readily in the circulation, and these will become emboli, and the plugging of the small arteries may lead to their aneurysmal dilatation and rupture of the vessel.

(vii.) This mention of the purpura allied to the increase of the colourless corpuscles leads us to the hæmorrhages seen with enlarged spleens. The conjunction of an enlarged spleen with epistaxis was repeatedly noticed by Hippocrates,[1] and in our day Virchow has specially insisted on the concurrence of an enlarged spleen with the hæmorrhagic state;[2] Dr. Habershon has also pointed out how commonly the spleen is enlarged in cases of purpura.[3]

Purpura is associated with many diseases in which a large spleen is seen; all the fevers, including the agues, and syphilis, (I believe that Dr. Gee was the first to point out how often the spleen in syphilis is enlarged,) cirrhosis of the liver, and many others. It thus becomes hard to say what is the precise relation of the enlarged spleen to the hæmorrhages.

(viii.) Dr. Hilton Fagge has drawn attention to the association of purpura with multiple sarcomata.[4] It has long been known that rapidly growing tumours often show hæmorrhages in their substance, and this has been explained by the tendency to rupture of all newly-formed vessels, whether in the fœtus, suppurating granulations, or the fungus hæmatodes.

(ix.) Then follows the purpura seen in some cases of rheumatism, to which Schönlein drew attention under the name of peliosis rheumatica.[5] I cannot think that there is any special variety of purpura in the case which I am about to relate; it

[1] Hippocrates, Epidemics, Book iii. Littré's ed. t. iii. p. 121, and Epidemics, Book ii., Littré's ed. t. v. pp. 87 and 95.

[2] Virchow, Allg. Störungen der Ernährung und des Blutes in Handb. d. spec. Path. u. Ther. Erlangen, 1854, p. 247.

[3] S. O. Habershon, Guy's Hospital Reports, 1857, p. 89.

[4] Hilton Fagge, Guy's Hospital Reports, 1881, p. 1.

[5] Schönlein, Allgem. u. spec. Path. u. Ther. Freyburg, 1837, Bd. ii. p. 45.

would seem to be allied to, if not identical with, erythema nodosum or hæmorrhagicum, and the purpura urticans of Bateman.[1] As long ago as 1824, Samuel Plumbe asserted that erythema nodosum was nearly allied to purpura.[2] It might be that this rheumatic purpura is allied to the toxic purpuras. Acute rheumatism is thought by some to be caused by the appearance in the blood of poisonous waste products, and they appeal to the appearances caused by lactic acid as proof of their views.

(x.) After having excluded all the foregoing states, there will remain a certain number of cases of purpura; not so many as might at first sight be thought, which cannot be ranged under any of these heads, and which must therefore be called idiopathic purpura. Of these cases the pathology is, if possible, more obscure than those which have preceded it.

Such being the various clinical states in which purpura is seen, what are the appearances seen after death which may reasonably be set down to having a share in causing the purpura? There are records of careful examinations after death, some of the later ones made with all the appliances that modern histologists demand, and yet no changes have been discovered either with the naked eye or with the microscope. It is possible that this may be due to imperfect methods. For how long was the spinal chord in locomotor ataxy and other nervous diseases affirmed to be unchanged? Yet so competent an observer as Hayem described in one case marked changes in the vessels, while in another which he saw he was forced to own that nothing unnatural could be discovered.[3] And of the changes which have been found, it can hardly be said that they give any ready explanation of the cause of the hæmorrhages.

Dr. Wilson Fox was the first to point out any changes in the histology of the vessels in purpura. He found both the small arteries and the capillaries in the neighbourhood of the purpuric spots (but not in the spots themselves) to stain with iodine, the supra-renal capsules, the stomach and intestines, being also markedly amyloid. Dr. Wilson Fox also noticed certain points which have been strangely neglected by those who have followed him, but which seem to me to be of considerable importance: (1.) The presence in the blood during life of a great excess of white corpuscles, followed in a few days by a diminution of the excess, while the corpuscles themselves became very granular,

[1] Thomas Bateman, A Practical Synopsis of Cutaneous Diseases, London, 1813, p. 115.

[2] Samuel Plumbe, Practical Treatise on Diseases of the Skin, London, 1824, p. 375.

[3] Ruc, Union Méd. 1870, t. ix. série iii. p. 680.

Dilatat capillaires Progres. med. 1884
p. 457.
Arnozan Arch de phys. normale 1883.
Cornil & Rigal Memoire de la
Société méd. des Hôp. de Paris
Année 1879. p. 75.

some distinctly disintegrating, and some granular *débris* were seen in the field, corresponding in all essential characters to the granules seen in the cells; (2.) the trouble with which the purpuric limbs could be injected; and (3.) the appearance of an increase in size and number of the nuclei of the adventitia of the vessels in scattered spots and patches, chiefly in the neighbourhood of the extravasations.[1] It must be added that Dr. Wilson Fox's patient had contracted syphilis six months before his death, and that this constitutional disease was apparently the cause of his death.

Now the multiplication of the nuclei of the sheath of the vessels, spoken of by Dr. Wilson Fox, is a very important change. It is closely akin to the inflammatory processes which have been described by some other authors. Hayem has recorded three cases of purpura accompanied by multiplication of the endothelia of the artery and by obliteration of the vessel, to which he gives the name of hyperplastic endarteritis.[2] Max Zimmermann has found the walls of the arterioles, not of the arteries, thickened to twice their natural size, the greater part of this thickening being due to the increase of the adventitia, which was full of young cells; while in opposition to the appearances seen in Hayem's cases, the inner coat was hardly at all changed, and the capacity of the vessel unaltered. Many of the vessels in the neighbourhood were, however, obliterated.[3]

The last of Hayem's cases also presented another resemblance to Dr. Wilson Fox's in the presence in the blood of an excess of white corpuscles, a large number of which appeared to be young, smaller than usual, and with one large nucleus, while others were larger than natural and held two or three nuclei. Hayem is inclined to think that the white corpuscles may form into emboli, and thus lead to infarctions.[4] One of the results of emboli should be kept in mind, viz., that aneurysms may follow;[5] and this indeed has been seen in a case examined by Hayem and recorded by

[1] Wilson Fox, British and Foreign Medico-Chirurgical Review, 1865, vol. xxxvi. p. 480.

[2] Hayem, Comptes rendus des Séances de la Société de Biologie, Année 1870, p. 24, and Année 1876, p. 232.

[3] Max Zimmermann, Arch. d. Heilkunde, 1874, Jahrg. xv. p. 166.73

[4] Panum found numerous ecchymoses as the result of embolism experimentally induced. (Arch. f. Path. Anat. 1862, Bd. xxv. p. 513.)

[5] I should like to say that this sequel to emboli had been pointed out by Dr. John W. Ogle (Transactions of the Pathological Society of London, 1857, vol. viii. p. 168, and Med. Times and Gazette, 1866, vol. i. p. 196), by Mr. Jolliffe Tufnell (Dublin Quarterly Journal of Medical Science, 1853, vol. xv. p. 371), by Mr. T. Holmes (System of Surgery, London, 1862, vol. iii. p. 353), and by Dr. Church (these Reports, 1870, vol. vi. p. 99), some years before Ponfick's paper in Virchow's Archives. (1873, Bd. lviii. p. 557.)

Huchard.[1] Numerous miliary aneurysms were found in the brain in the midst of hæmorrhagic points, and the walls of the vessels showed a marked "cellular irritation," granular infiltration, and fibroid transformations. These changes chiefly involved the sheaths. The woman was only 32 years of age: no like changes were seen in the heart, but no other organ seems to have been examined for vascular changes.

It seems quite possible, if there be any truth in the doctrine of aneurysm from embolism, that the infarction of plugs formed of the colourless corpuscles (which in some cases of purpura, at all events, are in excess) may lead to miliary aneurysms such as were found in this woman's brain, and which in her seem to have caused the multiple ecchymoses of that organ. It is much to be regretted that no other organ but the heart was examined. Her age would hardly be advanced enough to cause the aneurysms which have been so well described by Charcot and Bouchard.

The multiplication of the nuclei of the sheath of the vessels has been seen in other hæmorrhagic diatheses, viz., in the persistent and congenital hæmorrhagic diathesis of hæmophilia. The following case of Lindwurm's resembled Dr. Wilson Fox's in two particulars—the appearance of amyloid degeneration and in multiplication of the nuclei.

The cutaneous vessels of a bleeder, who had never had syphilis, and whose skin, from the crown of his head to the sole of his foot, showed not a single sound place, nothing but patches of pityriasis rubra, lichen ruber, eczema squamosum, ichthyosis, and fifty to sixty ulcers, were examined by Buhl. Besides a thickening of the papillæ and their epidermis, he found an overgrowth of the capillary vessels, and with this an increase in number of the nuclei of the capillaries, which also were readily stained by iodine.[2]

So also in Dr. Percy Kidd's case, the vessels all showed marked proliferation of the walls.[3] But I am bound to say that since Dr. Kidd published his case, I have examined the tissues in two cases of hæmophilia, and have been unable to detect any changes whatever in the vessels.

In scurvy there is a hæmorrhagic diathesis, and Uskow, about five years ago, described the vascular changes in the gums: the vessels are full of red or white corpuscles, apparently a thrombosis; the "lumen" may be greater than natural; where the white corpuscles prevail, there the cells of the endothelium

[1] Huchard, Bulletins de la Société Anatomique de Paris, 1870 (xlv. année) p. 172.
[2] Lindwurm, Zeitschrift f. rat. Med. 1862, Bd. xiv. p. 263.
[3] Percy Kidd, Med. Chir. Trans. 1878, vol. lxi. p. 243.

Keller, Revue de med. Aug. 1884. (abst. in Med. Chron. Oct 1884. p. 48.

are swollen, but they were never seen proliferating; afterwards "fibrous bands" formed around the vessels, with "granulating cells" between them. The number of red corpuscles not altered, nor of white.[1]

In Ducastel's case of purpura, which was examined by Cornil and Frémont, there was found in a patch of purpura of the lower lip great dilatation of the vessels of the papillæ, fifteen to thirty times greater than natural, the connective tissue being thinner and more delicate than usual in the papilla. Throughout the patch of purpura there was a good number of emigrated leucocytes, but no red corpuscles.[2] This last appearance is hard to understand, and makes it doubtful if the patch were really purpura. In the skin, however, were found no such changes in the vessels, though here red corpuscles were discovered around them. Nowhere could any arteritis or periarteritis be found.

It will thus be seen that histology has but little help to give us in understanding the process of purpura. The change which is most constant, the overgrowth of the cells of some part of the wall of the vessel, does not readily explain an easy rupture of the wall. It may be noted that many of the causes of purpura are toxic in their source, such as the purpura seen after iodide of potassium, in poisoning by phosphorus, and in chronic jaundice. Had I been more successful in causing hæmorrhages in the web of the frog's foot after the injection of the iodide of potassium and other alkaline salts, I should have been more ready to set down all the toxic purpuras to the transpiration of the red blood corpuscles; but as it was only under the action of one salt, chloride of sodium, that I saw the red corpuscles transpire, I feel some hesitation in thinking that all the toxic agents which I have named cause hæmorrhage *per diapedesin*.[3]

Of late years there has been made a determined attempt to revive the old doctrine of Stahl, that the soul, or in modern language, the nervous system, is the frequent cause of hæmorrhage. The arguments in favour of this explanation of purpura have been lately urged with much force and ability by Dr. Stephen Mackenzie,[4] but I do not think that the evidence is any stronger, either from physiological experiments or from

[1] N. Uskow, Centralblatt f. d. m. W. 1878, p. 498.

[2] Du Castel, Des diverses Espèces de Purpura, Thèse de Paris pour l'Agrégation, 1883, pp. 56–58.

[3] Stroganow (Arch. f. Path. Anat. 1875, Bd. lxiii. p. 540) has described the presence of red corpuscles between the coats of the aorta and other vessels in a woman who died soon after a hard labour, but other morphological changes were not seen. He also makes the following statement, which seems strange after Dr. Wilson Fox's and Hayem's careful observations: "Wir fanden in der Literatur keinen sorgfältig, histologisch-untersuchten Fall von Werlhoff'scher Krankheit."

[4] Stephen Mackenzie, British Medical Journal, 1883, vol. ii. p. 409.

clinical observation, than when I examined the subject more than ten years ago.[1]

Setting aside the demonstration of an increase of the colourless corpuscles in the blood in some cases of purpura, there seems but little to record of any increase of knowledge of the state of the blood in this disease. The instrument for numbering the corpuscles in a cubic millimeter is a distinct help to the clinical physician; but in purpura it has only told us what we might have looked for, viz., that the red corpuscles were diminished. The chemistry of the blood, like so many other parts of physiological chemistry, is in too imperfect a state to give us at this moment any aid.

Let us turn now to the details of our case:—

Charles D., aged 18, admitted into Luke's Ward on February 3, 1883.

For the greater part of these careful clinical notes I am indebted to Mr. Oswald A. Browne, the house-physician, though in some places I have added a few observations of my own.

He is a patent-capsule-maker, and much tin is used in his trade. He says that he is a teetotaller, and lives at Holloway, where there is no illness in the same house or street. He was vaccinated when a baby, but not since.

He has been well fed, and not lacked meat or vegetable food.

There is no family history of bleedings or of purpura on the father's or mother's side.

He had rheumatic fever twelve years ago, and again in May last, when he was in University College Hospital; and for the following information as to his sojourn in this hospital I am indebted to my friend Dr. Thomas Barlow. He was in the hospital from May 23 to July 8, his illness having begun three weeks before admission. He had a presystolic and systolic murmur at apex and a systolic murmur at the base; no skin disorder was noted. His temperature varied from 99° to 100°; rarely more than this. There were enlargement of the knuckles and swelling of the elbows: the other joints were also stiff and somewhat swollen.

He has had what he calls rheumatism every winter for the last eleven years, and he was then usually laid up for six or seven weeks. In none of them did he have any spots on the body, as in the present attack. He has never had hæmoptysis or hæmaturia.

He denies syphilis, but admits having had a running; he lived and worked among loose companions.

The present illness began about a fortnight before February 1.

[1] See my Treatise on Hæmophilia, London, 1872, p. 94.

He complained of pains in both legs below the knee and pains in the shoulders. On January 28, 29, and 30, the pains were increasing, and on the 30th he was seen by Mr. Whittingham of the Holloway and North Islington Dispensary, who tells me that he found him lying in an underground back-kitchen, the front room of which was used as a laundry. On this day Mr. Whittingham says the rheumatic symptoms were not so severe, but the dyspnœa from the heart-disease was prominent. A mixture containing three grains of iodide of potassium at each dose, with solution of acetate of ammonia, nitric æther, and aromatic spirit of ammonia, was ordered to be given every four hours. No hæmorrhagic spots were seen on January 30, or on February 1, on which day he appeared somewhat relieved. But in the afternoon of this day the patient noticed several bruises on his left elbow, and these quickly spread—so quickly that his mother assured me that one could see them growing after they had come out. The same "bruises" also were seen on both arms, the back, and both hips. He said that these bruises began as small red pimples, which rapidly increased in size and took on a dark colour.

He has had no shiverings, sickness, or pain in the back.

Present State.—A lad with light hair and eyes, attention being at once drawn to the coloured patches on the face. Scattered thickly over the forehead and cheeks, not more on one side than the other, are raised indurated patches, for the most part circular, the size of a threepenny-piece, some smaller. Most of them are of a deep mulberry colour, not changed by pressure, with hardening of the skin and subcutaneous tissue for some distance beyond the margin of the coloured patches. There are some small raised patches of a rosy colour, also firm.

Both eyelids are swollen and show purple hæmorrhagic mottling under skin; the conjunctivæ are quite free from hæmorrhage though suffused. With the ophthalmoscope nothing unnatural can be made out in either eye.

Both alæ nasi are covered with mottled patches, varying in colour from a bright red to deep mulberry tint; the patches being very firm and the thickening extending into the tissues for some distance beyond the margin of the patches. Both lips are much swollen, shiny, everted, coloured in same way; the mucous membrane between the patches being softer and almost white. The gums are not swollen, but are very dark, apparently from hæmorrhage along the margin of teeth. Breath foul. Tongue swollen, slightly hardened, of a bright red colour, with slight fur on dorsum. The uvula and the mucous membrane of the palate and fauces almost black, apparently from presence of

blood underneath. Voice husky, and complains of some difficulty in swallowing.

The entire chin is considerably indurated and covered by one large patch of subcutaneous hæmorrhage, dark mulberry in colour.

Both ears are swollen, tense, shining, firm; here and there, on both surfaces, ecchymoses.

On the front of the neck are a few small subcutaneous hæmorrhages, about the size of peas, of a brighter colour, but also raised and firm. (A water-colour drawing of face is in the Museum.)

On front of chest and belly also smaller and brighter patches circular in shape, but very discreetly scattered and not numerous.

The right arm has also raised hard dark purple patches, scattered thickly over it on the extensor surface; only here and there on the flexor surface. Many are nearly as large as a shilling-piece, for the most part circular, but some are confluent into large patches. The induration of the tissues around these patches is also here very noticeable. The smaller spots here are of a bright red colour.

On the head of the right ulna is an indurated oval patch, mottled at its centre, and for the rest bright red, the margin of induration being distinctly marked by a white line.

The metacarpo-phalangeal joints of the first three fingers are much swollen and deformed, and there are small hæmorrhagic patches like the others over the backs of the first two of them.

Nails natural. Palmar surface of both hands free.

The whole of the left arm is swollen and very tender, the extensor surface of the arm above the elbow being occupied by one large irregular-shaped patch of a deeper colour than elsewhere (and deeper in some parts than in others) everywhere raised, hard, very tender, and pitting on pressure. There are also several large patches on extensor surface of fore-arm, one large patch taking up the whole of the middle third of that surface, and extending widely over the inner surface of the fore-arm, the whole pitting on pressure and very tender.

Over the upper half of the back are several discrete smaller patches like those already described. The lower half shows a large patch, almost square, about 8 inches by 8 inches; here are some ecchymoses almost linear. The buttocks, crest of the ilia, and extensor surfaces of legs have the same kind of patches as elsewhere, but they vary more in size and colour. The soles of the feet are free. The great toes are directed outwards at an angle of 45° to the line of the metatarsal-phalangeal joints.

The penis shows a few hæmorrhages. There is nothing noticeable on glans.

He denies any sensation of itching in the skin.

Liver and spleen not palpable.

Heart: Apex beats in the fifth space in nipple-line. The area of dulness unchanged—query, a thrill perceptible at apex. Here is a prolonged murmur followed by a sound. The latter part of murmur and the impulse are synchronous. At base there is another murmur with first sound, distinct from that at apex, heard loudly over the middle of sternum and conducted up to the second right costal cartilage.

Lungs free, save a little fine crepitant râle at each base.

Blood from finger shows no increase of white corpuscles; the red show a marked tendency to form rouleaux. No bacterium seen.

Urine acid, sp. gr. 1030. A distinct trace of albumen; a cloud of mucus, but no smoky appearance.

Temperature on admission, 98°; at 2.30 P.M., 101°; at 8 P.M., 101.6°.

Feb. 4.—Last evening he was removed to an isolation ward. He has slept well, not delirious, and taken food well. Pulse small, regular, and compressible. Bowels open twice, stools natural, no blood. Looks much better and brighter. All over the body the colour of the patches is fading, and they are not so firm and raised as yesterday. The lips and ears are both less swollen and tense. The left arm swollen above and below elbow, shining, pitting on pressure, and very tender. Over the patches about both elbows distinct bullæ.

Heart as yesterday. Mr. Browne felt a very fine vibratile thrill at apex, and noted at apex a blowing murmur and a sound. The murmur probably presystolic and systolic. At base a well-marked blowing systolic murmur, heard most loudly over junction of second and third costal cartilages.

Urine shows a trace of albumen with abundance of pink urates.

Temperature at 2.30 A.M., 100.4°; at 9 A.M., 100.2°.

Evening.—Pulse very small and weak; complains of head feeling tight and of general aching.

Temperature at 8 P.M., 102.6°. Quinine to be omitted.

Feb. 5.—Has slept well during night and taken food well.

Temperature at 2 A.M., 101.4°; at 9 A.M., 100.8°. Pulse 110.

Colour in the blotches everywhere fading. Feels better. Urine still shows a trace of albumen.

The blood was examined for bacteria to-day by Dr. Vincent Harris, who has paid much attention to the detection of these organisms in the blood. His observations give a negative result, as the following report from him shows:—

"The blood was examined after being stained with a 0.1 per cent. aqueous solution of methyl-violet, dried on covering glass, and mounted in Canada balsam. The coloured corpuscles showed a marked tendency to run together, but were to all appearances otherwise normal. The colourless corpuscles were in slight excess, but presented no abnormality in structure. There was no indication of the presence of any form of bacteria in the specimens examined."

Temperature at 3 P.M., 101.8°; at 8 P.M., 101°.

Feb. 6.—The colour continues to fade, though on the neck and back are several fresh circular patches raised and indurated like those described on February 4. The arms are less swollen. He takes food well. Pulse 120, regular, and of fair volume. Urine, sp. gr. 1031, and with a trace of albumen.

Evening.—Pulse 92, regular, soft.

Temperature at 4 A.M., 100.2°; 9 A.M., 100.8°; 8 P.M., 102.2.°

Feb. 7.—The patches are almost confluent over the face, neck, and shoulders, and are of a deep mulberry colour. Elsewhere the colour is fading; now is of a light brick-red; most of the patches are very tender. Lips and ears less swollen, but the gums are now tender, and the tongue has a thick, white, moist fur.

Temperature at 8 A.M., 100.4°; 8 P.M., 100.6°.

Feb. 8.—He slept fairly. Not delirious.

Temperature at 8 A.M., 99.4°; at noon, 102.2°; at 7 P.M., 102.4°; at 10 P.M., 102.4°.

The purpura has much spread over neck and upper part of chest and back; it is of a deep mulberry colour; not so raised or so indurated as on admission. Some of the earlier patches have quite faded. Pulse 120, small and soft. He takes fluids well. Tongue thickly furred and the breath very offensive. The uvula and the right tonsil are not enlarged, and they show a light brown appearance—query, due to a slough of mucous membrane. Rest of throat reddened. Penis is clubbed from a large hæmorrhage into foreskin covering glans. The skin here looks ready to slough.

The ophthalmoscope to-day detects nothing abnormal.

Feb. 9.—Purpura again spreading. He still sleeps and takes food well. Large bullæ over the left arm.

Temperature at 8 A.M., 101.6°; 1.30 P.M., 101°; 8 P.M., 102°.

Feb. 10.—Restless night; talking much, but appeared rational when spoken to by nurse.

Temperature at 8 A.M., 100.8°.

Over lower part of both lungs a good deal of râle. Uvula distinctly sloughing; the left side of palate also white. Tongue

thickly furred and breath very offensive. Further fresh patches of purpura, so that back is almost covered by them. Pulse 120, soft and compressible. Urine 1034, and with a well-marked trace of albumen.

In the afternoon the breathing became more rapid; respiration 60. He spat up bloody-coloured sputa. His mental state was clouded. The râles in the chest were now audible over a larger extent of surface, and were looser in character. He died at 9.30 P.M.

Examination thirty-seven hours after death, 10.30 A.M., *Feb.* 12, 1883.—Rigor mortis well marked; the body thickly set with purpuric spots; the patches are confluent over the chest; the penis is clubbed from hæmorrhage into the foreskin.

On dividing the scalp, symmetrical hæmorrhages are seen between periosteum and skin over each ear and eye.

The calvaria is natural; the meninges, vessels, sinuses, and the substance of brain are quite natural. No hæmorrhages are seen anywhere within the head.

The mucous membrane of the lips and gums has sloughed at the spots where the hæmorrhages were seen during life. The velum is much thickened; the forepart of the uvula is covered with a thick brown slough, which cannot be torn off or detached by washing; the right tonsil is much enlarged and sloughing; the left is natural. The back of the uvula still shows hæmorrhage, and the back of velum shows a slough the size of a shilling. The gullet is natural; shows no hæmorrhages. The epiglottis shows a hæmorrhage at its tip and below; the rest of the larynx and trachea is natural. The glandulæ concatenatæ are much enlarged.

On opening the chest, there is seen to be a considerable excess of fluid in both pleuræ, which are studded with ecchymoses; a few old adhesions on the right side at back; there are more on left side, into one of which a hæmorrhage has taken place.

The pericardium is universally adherent by old adhesions. There is a calcareous patch over the left auricle. The right auricle is much dilated. There is no clot in the auricular appendix. The tricuspid orifice is not contracted; but the valves show a milk-white appearance, and there is a small hæmorrhage into the texture of the valves. The two anterior flaps are joined together, and there is a fringe of small granulations around the valve a little above the insertion of the chordæ tendineæ. There are numerous small ecchymoses in the endocardium of the conus arteriosus. The pulmonary valves are natural. The left auricle is much dilated. There is no clot in the auricular appendix. The mitral orifice grasps tip of middle finger, and a

row of small granulations can be seen from above. On opening the ventricle from below, the usual funnel-shaped appearance of mitral constriction is seen, and the chordæ tendineæ are thickened and opaque. Fine ecchymoses are seen over the endocardium. The aortic valves are much deformed, joined together, and a crown of granulations surrounds their ventricular surface. There is some hypertrophy and dilatation of the left ventricle, but the muscular tissue is of a brownish pale colour. The weight of heart is 575 grammes. Before opening the heart, water poured into the aorta slowly ran through into the ventricle.

The bronchial tubes contain bloody mucus. The large pulmonary arteries are free. The upper lobe of the right lung is solid, sinking in water, red, highly granular on section. The lower lobe shows same appearances, but its colour is more purple, and distinctly suggests hæmorrhage. The same appearances are seen in the lower lobe of the left lung, but with grey-coloured, granular, irregular-shaped masses about the size of peas inserted in the purple tissue. The upper lobe of left lung is œdematous.

There are hæmorrhages into the peritonæum covering the small intestines and the recti muscles.

The spleen is large, firm, natural in pulp and follicles, weighing 250 grammes.

The small intestines are natural on their mucous surface. The large intestines show four ulcers. The largest is just below the rim of the ileo-cæcal valve, causing this to be swollen. It is irregular in shape, but could be covered by half-a-crown. A large slough is still adherent. There is no hæmorrhage around. In the ascending and transverse colon are three ulcers the size of sixpenny-pieces, but slightly oval, surrounded by hæmorrhage, and the sloughs are still adherent.

In the stomach there are some few ecchymoses in great cul-de-sac. The pancreas and duodenum are natural.

The liver is natural in size. On section it shows a distinct nutmeg appearance. The tissue is not increased in firmness. The gall-bladder is collapsed; there is a very little yellow bile in it.

The kidneys show a slight amount of cloudy swelling; weight, 400 grammes together. There are no hæmorrhages in the pelvis.

Aorta natural—query, slight atheroma.

The two elbows, knees, and ankle-joints opened. No hæmorrhages found. An increased quantity of a yellow synovia in left ankle, with shreds of fibrine.

Small portions of the mucous membrane of the lips and gums, of skin over the pectoral muscles and abdominal recti, and of the kidney, were hardened in chromic acid and spirit, and sections

examined in the month of March. Attention was of course specially directed to the vessels, but I could find no changes either in the mucous membrane or in the skin, though portions of the skin were the seat of abundant hæmorrhage. They were not examined for amyloid reaction until the month of November, when Dr. Vincent Harris very kindly undertook to take them out of the Canada balsam and test them for amyloid disease. He writes to me: "On examination of the specimens, my opinion is, that they are not amyloid. I am not able to find any abnormality in the blood-vessels. I fancy the larger vessels of the kidney are rather too well supplied with muscle, but it may be only fancy; at any rate, there is no gross change."

When the lad was brought by his friends to St. Bartholomew's Hospital, I felt that the question of some acute specific hæmorrhagic affection was one which had to be discussed. He had fever and pains in the joints, together with abundant purpura. It was Saturday, and the mother then told us that the purpura had appeared on the Wednesday. The condition of the patient, still fair on the fourth day of the disease, made us think that the purpura could hardly be due to a hæmorrhagic form of small-pox or other fever. Then was it some form of a toxic purpura? He worked in tin and other metals, and two days before the appearance of the purpura, a mixture containing three grains of iodide of potassium for a dose had been given every four hours. But I doubt if the iodide rash be ever so severe or so general as in this case. It is petechial, and more limited to the lower limbs. Then as to the morphological elements of the blood, neither I myself nor the skill of Dr. Vincent Harris could detect during life any marked increase of the white corpuscles, or any signs of the presence of bacteria. Nor could bacteria be found after death in the purpuric tissues. So that at least in one case of fatal purpura the *Monas hæmorrhagicum* of Ceci has not been found, even though carefully looked for. The liver and spleen could not be felt, but there was evidence of disease of the mitral orifice; and there was proved, by the examination after death, to be great deformity of the aortic valves with stenosis of the mitral orifice and disease of the tricuspid orifice, illustrating a conclusion which may I think be drawn from Dr. Bedford Fenwick's paper,[1] that disease of the tricuspid (when it is seen) often accompanies mitral disease. Was then the purpura due to the cardiac disease congestive in its origin, or due to numerous emboli? Against the origin of the purpura in embolism is the fact that the spleen and the kidneys were free from

[1] Bedford Fenwick, Transactions of the Pathological Society of London, 1882, vol. xxxiii. p. 64.

infarctions. Had these large spots of purpura been due to embolism, the emboli must have been tolerably large—large enough for their fellows to have caused effects visible to the naked eye in the spleen or the kidney, which could hardly have escaped in so general a diffusion. In fact, there was no appearance of embolism found in the body, if we except the patches of purpura.

Was the disease scurvy? During life this was an opinion held by some, and they pointed to the state of the gums as proof. But it must be owned that swollen and bleeding gums are not uncommon in cases of hæmorrhagic diathesis in which imperfect diet cannot be traced, and in which other recognised causes are present. This lad, we were told, had had a due supply of fresh vegetables and flesh-meat.

Finally, were there present any of the other causes of purpura spoken of at the beginning of the paper? I could not find certain evidence either before or after death of such constitutional states as hæmophilia, syphilis, leucæmia, Bright's disease, jaundice, phthisis, and the like. But there was a marked history of rheumatism—rheumatism which had begun when he was only six years old, and had returned every winter, and from an acute attack of which he had suffered only the May before his death. Further, the purpuric patches were for the most part raised, hard and tender, calling to mind at once patches of erythema nodosum; and they took up a like place on the extensor surfaces, what may be called the psoriasis position, leaving the palms and soles free. The patches were also free from itching, in this way showing another point of resemblance to the purpura urticans or erythema hæmorrhagicum of our own day; they faded and became less raised with a degree of quickness not often seen in ordinary purpura.

I incline therefore to the opinion, that the case which is now printed is one of rheumatic fever accompanied by purpura, to which the name of rheumatic purpura may very well be given. I do not see how a line can be drawn between a case like this and those which Schönlein described under the name of peliosis rheumatica.

The word peliosis is used by Hippocrates,[1] and is apparently derived from πελιός, discoloured by extravasated blood. The same spirit, which now tempts every good dermatologist to change all his names for diseases about every five years, moved Schönlein, fifty years ago, to substitute peliosis for purpura. He speaks of peliosis Werlhofii,[2] peliosis senilis, and the like.

[1] Hippocrates, De Fractis, 11 (of Calcaneum), Littré's ed. t. iii. p. 456.

[2] Schönlein, Allgem. und spec. Pathologie und Therapie, Freiburg, 1837, Bd. ii. p. 45.

Caesar Boeck, Vierteljahrs. f. Derm. u. Syph.
1883. S. 481. Exanthema multiforme
& purpura rh. after pharyngitis.

In the same chapter he also spoke of a peliosis rheumatica, the description of which I will give in his own words: "The patches never run together, as they often do in morbus maculosus Werlhofii; the patients have either suffered before from rheumatism; or rheumatic symptoms appear at the same time; pains in the joints, which are swollen and painful on movement. The patches peculiar to the disease appear in the majority of the cases first in the limbs, especially on the lower, and here only below the knee. The spots are small, the size of a lentil (*Linse*) or a millet-seed, bright red, not raised, disappearing on pressure with the finger, becoming later on dirty brown yellow. The eruption appears in crops, and often lasts through many weeks. Slight changes of temperature will bring out fresh patches. The disease appears with fever which has a remittent type."

It will be seen that the case now printed agrees with Schönlein's description of peliosis rheumatica in all but the physical appearances of the patches. They were not small, but in many places confluent; they were raised above the skin, and firm, and they did not disappear on pressure; but are these differences enough in themselves to create a new species and to separate the case from those described by Schönlein? I think not, though I have known some Germans refuse the name of peliosis rheumatica to a generally diffused purpura in rheumatic fever, the size of the patches of which certainly exceeded a millet-seed, being as big as a sixpence. But to avoid using a new name, in itself not one whit better than purpura, I prefer to call the disorder rheumatic purpura.

Since the discovery that effusion of blood into the joints was the chief cause of the "rheumatism" in hæmophilia, and Scheby-Buch's observations on the presence of joint affections in purpura,[1] it may be asked: are hæmorrhages into the joints in cases of rheumatic purpura the cause of the rheumatic symptoms? This case does not give any affirmative answer to that question. The joints examined after death showed the appearances of ordinary rheumatic fever, but no hæmorrhages into their cavities. There had been many attacks of rheumatism before the hæmorrhage into the skin was noticed: indeed the friends denied any similar hæmorrhagic disease before the attack of which the patient died.

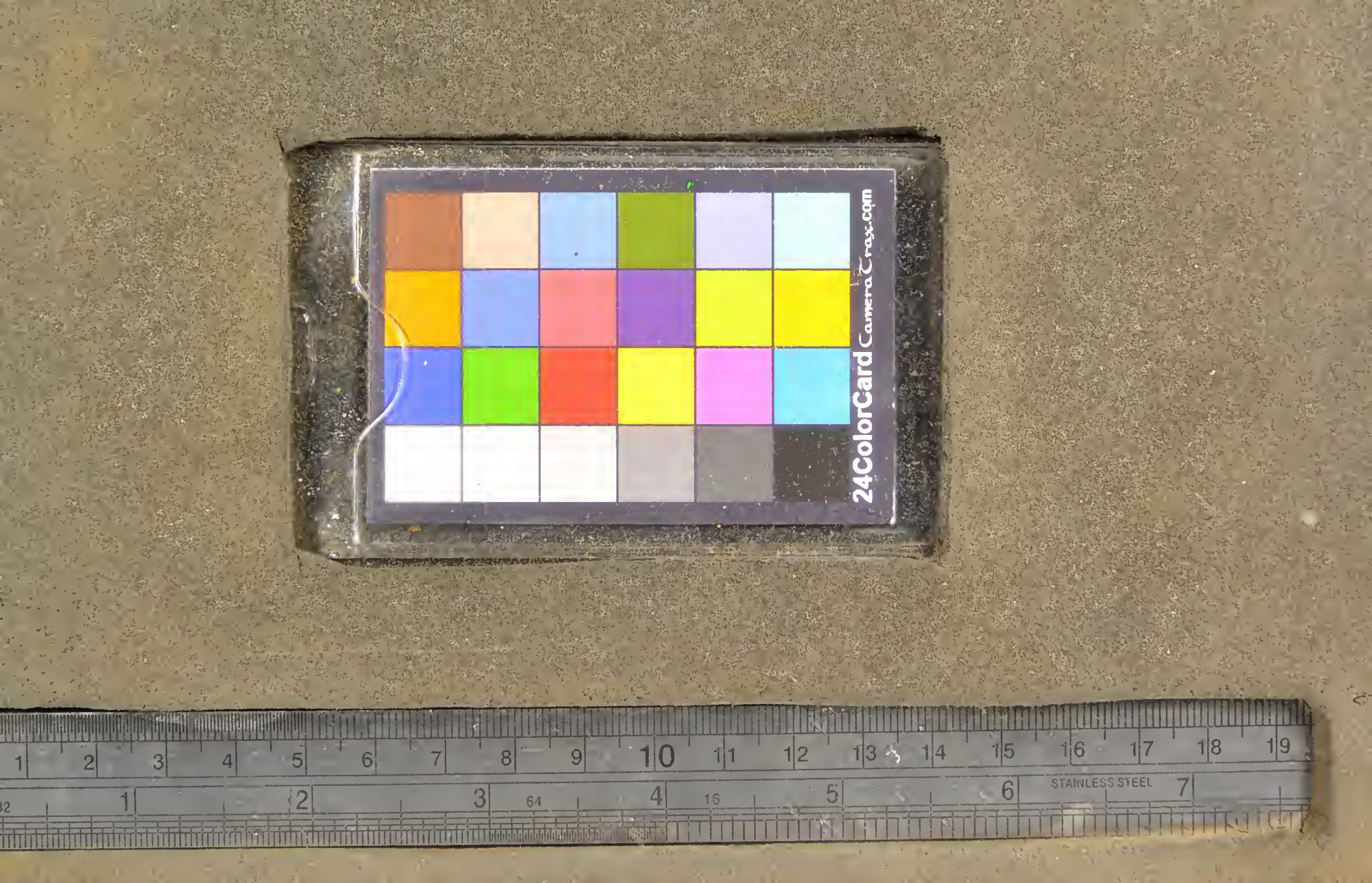
It may be noted that four ulcers of the colon were found. Ulceration of the intestines appears to be by no means rare in purpura. Max Zimmermann has recorded a case in which he found no less than 150 ulcers in the ileum, and the process of

[1] Scheby-Buch, Deutsches Archiv. f. klin. Med., 1874, Bd. xiv. p. 466.

inflammation had extended to the peritonæum.[1] Henoch has also spoken of colic in cases of purpura.[2] Are the ulcers merely the result of extravasation of blood under the mucous membrane, which then sloughs, and the slough is eaten off by the *succus entericus?* A process like this could be actually seen going on during life in parts exposed to view. For example, there was apparently a simple extravasation of blood under the healthy mucous membrane of the gums and uvula on the day of admission; before death the mucous membrane had sloughed, and it only needed time for large ulcers to form, and be like those in the intestine.

[1] Max Zimmermann, Arch. der Heilkunde, 1874, Jahrg. xv. p. 170.
[2] Henoch, Berlin. klin. Woch. 1868, p. 517, and 1874, p. 641.

Subord. Akten d. Ständetages p. 204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